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APPLICATION NO.	FILING DATE	FIRST NAMED INVENTOR	ATTORNEY DOCKET NO.
08/469,172	06/06/95	SEIDMAN C	IGI-111CN

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EXAMINER	
MYERS,C	
ART UNIT	**PAPER NUMBER**
1655	28

DATE MAILED: 03/27/01

Please find below and/or attached an Office communication concerning this application or proceeding.

Commissioner of Patents and Trademarks

Office Action Summary

Application No.	Applicant(s)
08/469,172	Seidman et al
Examiner	**Group Art Unit**
Carla Myers	1655

☒ Responsive to communication(s) filed on *Jan 5, 2001*

☒ This action is **FINAL.**

☐ Since this application is in condition for allowance except for formal matters, **prosecution as to the merits is closed** in accordance with the practice under *Ex parte Quayle*, 1935 C.D. 11; 453 O.G. 213.

A shortened statutory period for response to this action is set to expire *3* month(s), or thirty days, whichever is longer, from the mailing date of this communication. Failure to respond within the period for response will cause the application to become abandoned. (35 U.S.C. § 133). Extensions of time may be obtained under the provisions of 37 CFR 1.136(a).

Disposition of Claim

☒ Claim(s) *1-30, 32-34, and 36-46* is/are pending in the applicat

Of the above, claim(s) _____ is/are withdrawn from consideration

☐ Claim(s) _____ is/are allowed.

☒ Claim(s) *1-30, 32-34, and 36-46* is/are rejected.

☐ Claim(s) _____ is/are objected to.

☐ Claims _____ are subject to restriction or election requirement.

Application Papers

☐ See the attached Notice of Draftsperson's Patent Drawing Review, PTO-948.

☐ The drawing(s) filed on _____ is/are objected to by the Examiner.

☐ The proposed drawing correction, filed on _____ is ☐ approved ☐ disapproved.

☐ The specification is objected to by the Examiner.

☐ The oath or declaration is objected to by the Examiner.

Priority under 35 U.S.C. § 119

☐ Acknowledgement is made of a claim for foreign priority under 35 U.S.C. § 119(a)-(d).

☐ All ☐ Some* ☒ None of the CERTIFIED copies of the priority documents have been

☐ received.

☐ received in Application No. (Series Code/Serial Number) _____.

☐ received in this national stage application from the International Bureau (PCT Rule 17.2(a)).

*Certified copies not received: _____

☐ Acknowledgement is made of a claim for domestic priority under 35 U.S.C. § 119(e).

Attachment(s)

☐ Notice of References Cited, PTO-892

☐ Information Disclosure Statement(s), PTO-1449, Paper No(s). _____

☐ Interview Summary, PTO-413

☐ Notice of Draftsperson's Patent Drawing Review, PTO-948

☐ Notice of Informal Patent Application, PTO-152

--- SEE OFFICE ACTION ON THE FOLLOWING PAGES ---

1. This action is in response to Paper No. 27, filed January 5, 2001. Applicants arguments have been fully considered but are not persuasive to overcome all grounds of rejection. This action is made final.

2. The text of those sections of Title 35, U.S. Code not included in this action can be found in a prior Office action.

3. Claims 1-30 and 32-34, 36-46 are rejected under the judicially created doctrine of obviousness-type double patenting as being unpatentable over claims 1-5 of U.S. Patent No. 5,429,923. Although the conflicting claims are not identical, they are not patentably distinct from each other because the instant claims and the claims of '923 are inclusive of methods for diagnosing hypertrophic cardiomyopathy wherein the method comprises detecting the presence or absence of a hypertrophic cardiomyopathy associated mutation in the RNA of an individual. It is noted that the claims of '923 do not recite packaging the reagent required to perform the diagnostic method in a kit. However, reagent kits for performing DNA diagnostic assays were conventional in the field of molecular biology at the time the invention was made and therefore, it would have been prima facie obvious to one of ordinary skill in the art at the time the invention was made to package the primers and probes required for the detection of hypertrophic cardiomyopathy associated-mutations in a kit for the expected benefits of convenience and cost-effectiveness.

In the response of Paper No. 27, filed May 12, 2000, Applicants state that a terminal disclaimer will be filed upon indication of allowable subject matter if appropriate. Accordingly, the rejection is maintained for the reasons of record.

4. Claim 36 stands rejected under 35 U.S.C. § 102(b) as being anticipated by Eisenberg.

Eisenberg teaches RNA probes complementary to the sequences of *B*-MHC nucleic acids (see page 289). The probes are considered to have the property of being useful for facilitating diagnosis of hypertrophic cardiomyopathy because the probes of Eisenberg hybridize to and thereby are capable of detecting changes in the *B*-cardiac myosin heavy chain DNA. Accordingly, Eisenberg anticipates the invention of claim 36.

In the response of Paper No. 27, Applicants traversed this rejection by stating that Eisenberg teaches away from the claimed invention because Eisenberg teaches a probe that is not capable of distinguishing between alpha and beta myosin. It is stated that Eisenberg does not teach or suggest a probe useful for facilitating diagnosis of hypertrophic cardiomyopathy which is capable of detecting a hypertrophic cardiomyopathy-associated mutation. These arguments are not persuasive because the RNA probe of Eisenberg has the general property of being useful for diagnosing hypertrophic cardiomyopathy because the probe is capable of hybridizing to and detecting *B*-cardiac myosin heavy chain DNA. The claim does not recite any functional or structural language which distinguishes the claimed probe over those of Eisenberg. Any probe which hybridizes to beta-myosin heavy chain DNA would be useful in detecting a mutation in beta-cardiac myosin heavy chain DNA. For example, the probe of Eisenberg could be used in methods in which the hybridization conditions are controlled so that any single base change in the nucleotide sequence would be detectable. The claim does not require that the probe facilitate detection by any particular means and does not require that the probe distinguish between beta and alpha myosin. The RNA probe of Eisenberg would be expected to hybridize to the beta-

cardiac myosin heavy chain DNA since it is complementary to this DNAs sequence and thereby the probe could be used in, e.g. an enzyme digestion method to detect mutations in the DNA sequence. Applicants comments that the probe of Eisenberg does not distinguish between alpha and beta myosin chains is not relevant because there is no requirement in the claim that the probe has the property of specifically hybridizing to only beta- myosin and not hybridizing to alpha-myosin. The claimed probe is not defined with respect to any particular structural limitations which would distinguish the claimed probe over the RNA probe of Eisenberg. Because the claim is drawn to a product, there is no requirement that Eisenberg teach using the product for detection of hypertrophic cardiomyopathy. Rather, a proper rejection of the claim requires only a teaching in the art of a product having the same structural characteristics as the product claimed. In the instant case, the RNA probe of Eisenberg has each of the properties of the probe of claim 36, i.e., the probe comprises ribonucleotides arranged in a sequence which is complementary to at least a portion of the beta-cardiac myosin heavy chain DNA; the probe has the property of being useful for the diagnosis of hypertrophic cardiomyopathy; and the probe is capable of detecting a hypertrophic cardiomyopathy-associated mutation.

5. Claims 37 and 38 are rejected under 35 U.S.C. § 102(a) as being anticipated by Friedman.

Friedman teaches sets of nested PCR primers useful for the amplification of nucleic acids of *B*-MHC (see page 109). Because the primers of Friedman amplify nucleic acids of *B*-MHC, the primers have the inherent property of being capable of detecting mutations in the *B*-MHC gene, including hypertrophic cardiomyopathy-associated mutations. Accordingly, Friedman anticipates the invention of claims 37 and 38.

In the response of Paper No. 27, Applicants traverse this rejection by stating that Friedman does not teach primers useful for the diagnosis of hypertrophic cardiomyopathy by being capable of detecting a hypertrophic cardiomyopathy-associated mutation. It is asserted that Friedman teaches away from the claimed invention because the reference teaches that mutations could not be identified in exon 13 of patients with MHC. These arguments are not persuasive because it is a property of the primers taught by Friedman that they are useful in the diagnosis of hypertrophic cardiomyopathy because the primers are capable of amplifying the *B*-MHC DNA and thereby could be used for diagnostic analysis of the sequences of the amplified *B*-MHC DNA. Applicants appear to be asserting that Friedman must teach use of the primers for detecting hypertrophic cardiomyopathy mutations. However, the claims are not drawn to methods for detection of hypertrophic cardiomyopathy mutations, but rather are drawn to a set of primers. Again, it is a characteristic of the primers of Friedman that they are capable of detecting hypertrophic cardiomyopathy mutations. There is no requirement that Friedman teach all of the properties of the claimed primers. The issue is whether the product taught in the prior art is the same as the product claimed. In the instant case, the primers of Friedman are structurally and functionally indistinguishable from the broadly claimed primers.

6. Claims 37 and 38 are rejected under 35 U.S.C. § 102(b) as being anticipated by Feldman.

Feldman teaches compositions comprising sets of PCR primers useful for the amplification of nucleic acids of *B*-MHC (see page 1867). Because the primers of Feldman amplify nucleic acids of *B*-MHC, the primers have the inherent property of being capable of detecting mutations in the *B*-MHC gene, including hypertrophic cardiomyopathy-associated mutations. The

compositions of Feldman contain 13 pmol of each primer and therefore are considered to comprise at least 4 oligonucleotides. Accordingly, Feldman anticipates the invention of claims 37 and 38.

In the response of Paper No. 27, Applicants traverse this rejection by stating that Feldman does not teach a set of primers useful for facilitating the diagnosis of hypertrophic cardiomyopathy by being capable of detecting a hypertrophic cardiomyopathy-associated mutation. It is stated that Feldman evaluated gene expression in failing human heart, but doesn't teach detecting hypertrophic cardiomyopathy-associated mutations. Applicants arguments are not convincing because they are directed to limitations not recited in the claims. The claims are not drawn to methods for detecting hypertrophic cardiomyopathy-associated mutations. Rather, the claims are drawn to primers which have the property of being capable of detecting hypertrophic cardiomyopathy-associated mutations. In claims to products, if the product in the prior art is exactly the same as the product claimed, there is no requirement that the prior art teach each functional property of the product and there is no requirement that the prior art teach methods employing the product in the same manner that Applicants intend to employ that product. In the instant case, the primers of Feldman have each of the structural properties of the claimed primers. That is, the primers of Feldman amplify the MHC DNA and thereby are useful in facilitating detection of any disease, including hypertrophic cardiomyopathy, by being capable of detecting any mutation in the MHC gene, including hypertrophic cardiomyopathy-associated mutations. Applicant appears to be reading limitations into the claims. The claims do not require that the primers hybridize to a specific mutation, as with allele specific primers which amplify

only a mutated form of the sequence and not wild-type sequence, or vice-versa. Rather, the instant claims require only that the primers be capable of detecting hypertrophic cardiomyopathy-associated mutations and thereby the claims include any primers which amplify *B*-MHC DNA. Again, it is a characteristic of the primers taught by Feldman that they are useful in the diagnosis of hypertrophic cardiomyopathy because the primers are capable of amplifying the *B*-MHC DNA and thereby could be used for diagnostic analysis of the sequences of the amplified *B*-MHC DNA.

7. Claims 33-34 are rejected under 35 U.S.C. § 103 as being unpatentable over Geisterfer-Lowrance in view of Almoguera and further in view of the Stratagene Catalog.

Geisterfer-Lowrance teaches methods for detecting the presence of mutations associated with hypertrophic cardiomyopathy wherein the methods comprise detecting the presence of point mutations in the *B*-MHC nucleic acids by isolating DNA from individuals affected with hypertrophic cardiomyopathy and sequencing the DNA in order to identify the presence of mutations associated with hypertrophic cardiomyopathy (see, e.g., page 1000). In particular, Geisterfer discloses the presence of the missense mutation Arg403Gln and the association of this mutation with individuals having hypertrophic cardiomyopathy. Geisterfer-Lowrance does not teach detecting point mutations associated with hypertrophic cardiomyopathy by first amplifying sample *B*-MHC nucleic acids and performing a RNase protection assay.

Almoguera teaches methods for identifying gene mutations associated with genetically inherited diseases wherein the methods comprise amplifying a DNA sequence by PCR, combining the amplified DNA with a labeled RNA probe in order to form a RNA/DNA hybrid, and performing an RNase protection assay wherein cleavage of the RNA/DNA at regions that are not

hybridized as indicative of the presence of a disease associated mutation (see, for example, pages 39-41). In particular, the assay identifies single-base substitutions or point mutations which are considered to be "small alterations" in the DNA. Almoguera states that this provides a very rapid, efficient and sensitive means for detecting the presence of point mutations associated with diseases.

In view of the disclosure of Almoguera, it would have been obvious to one of ordinary skill in the art at the time the invention was made to have modified the method of Geisterfer-Lowrance so as to have detected the mutations associated with hypertrophic myocardiopathy in *B*-MHC nucleic acids by amplifying the nucleic acids by PCR and detecting the presence of mutations by performing an RNase protection assay using a labeled RNA probe in order to have achieved the expected advantages of providing a more rapid, efficient, and sensitive assay for the detection of hypertrophic myocardiopathy associated mutations in *B*-MHC nucleic acids.

Modification of the method of Geisterfer-Lowrance as discussed above would have resulted in a method for detecting point mutations in the *B*-MHC gene which required the use of the reagents of an RNA probe hybridizable to the *B*-MHC gene, PCR primers for the amplification of the *B*-MHC gene and a RNaseA for digesting unhybridized RNA. It is noted that at the time the invention was made the complete nucleotide sequence of the B-MHC was well known in the art and therefore the generation of primers and probes to perform the amplification/RNase protection assay of Almoguera would have been obvious to one of ordinary skill in the art and well within the skill of the ordinary artisan. The combined references do not

teach packaging these reagents required to practice the detection method or instructions for the detection method in a kit.

However, reagent kits for performing nucleic acid diagnostic assays were conventional in the field of molecular biology at the time the invention was made. In particular, the Stratagene catalog discloses the general concept of kits for performing nucleic acid detection methods and discloses that kits provide the advantage of pre-assembling the specific reagents required to perform an assay and ensure the quality and compatibility of the reagents to be used in the assay. Accordingly, it would have been prima facie obvious to one of ordinary skill in the art at the time the invention was made to have packaged the primers, RNA probe, and RNase in a kit for the expected benefits of convenience and cost-effectiveness for practioners of the art. Furthermore, it would have been further prima facie obvious to one of ordinary skill in the art at the time the invention was made to have included instructions in the kit in view of the conventionality of including instructions in kits for facilitating the use of the packaged reagents. It is noted that the written material in the instructions is not considered to be within the statutory classes and does not carry patentable weight (see MPEP 706.03(a)).

In the response of Paper No. 27, Applicants traverse this rejection by stating that Geisterfer-Lowrance only demonstrates that afflicted members of a single family with FHC have the mutation and that in the absence of extensive studies on the correlation between the mutation and MHC, one would not have a reasonable expectation of success for formulating the claimed kits. It is stated that at the time the invention was made it was possible that HC could be due to a point mutation at amino acid 403 of *B*-MHC, an alpha/beta cardiac myosin heavy chain hybrid

gene or a mutation that had not yet been described in the art. Applicants point out that the instant invention provides evidence that mutations can be used as an indicator of HCM. It is argued that there would not have been a reasonable expectation of success of the claimed methods or a suggestion for a collection of reagents in a kit. It is further stated that Geisterfer-Lowrance teaches away from the claimed invention because this reference states that since the mutation has been characterized in only two families, it cannot be predicted whether most individuals bear either of the two identified alleles, or whether the disease results from other new mutations.

Applicants arguments have been fully considered but are not persuasive because Applicants arguments are drawn to limitations not recited in the claims. Applicants state at page 11 that "the teachings of Geisterfer-Lowrance et al would not have suggested, to one of ordinary skill in the art at the time of the invention, the claimed methods for facilitating the diagnosis of hypertrophic cardiomyopathy". However, the claims are not drawn to methods. Rather, the instant claims are drawn to products. In claims to products, such as kits, the intended use of the product does not carry weight with respect to the obviousness of the product. While the teachings of Geisterfer-Lowrance may not have been sufficient to enable absolute diagnosis of HCM, the prior art suggests use of the disclosed sequences to amplify *B*-MHC nucleic acids and to identify mutations. Applicants arguments suggesting that the prior art must teach an unequivocal ability to diagnose HCM using primers is not appropriate for the instant rejection over claims drawn to kits. Again, the intended use of the reagents in the kit does not carry any weight with respect to the obviousness of the invention. Thus, the prior art when considered as a whole would have suggested the claimed kits for the benefits of convenience and cost-

effectiveness for practioners of the art wishing to amplify and identify mutations in the *B*-MHC nucleic acids. Applicants further state that they "traverse the Examiner's argument" that the kits provide the advantages of pre-assembly of reagents and reagent quality and compatibility assurance. It is stated that the kits of the present invention facilitate HC and thereby are suitable for diagnosis of FHC and SHC. These arguments are not convincing because the motivation provided by the prior art for obtaining the claimed invention need not be the same as that supplied by Applicants. Furthermore, the claimed kits have each of the structural limitations as the kits claimed by Applicants and thereby also have each of the properties of the kits claimed by Applicants. Applicants "traverse the Examiner's argument" that the instructions included as part of the kits are not statutory subject matter "as kits for diagnosis of human disease require approval of a governmental regulatory agency, including approval labels and other written materials". This argument is irrelevant to the issues at hand. The fact that regulatory offices, such as the FDA, require labels and other written materials does not in any way alter the fact that the U.S. Patent Office does not consider written material to be statutory subject matter. Again, Applicants attention is directed to MPEP 706.03(a), which states that " a mere arrangement of printer matter" is not within the statutory classes. Therefore, the printed material in the claimed kits does not carry weight with respect to the obviousness of the kit. Accordingly, Applicants comments regarding the fact that printed instructions would provide information as to how to analyze positive and negative results is not persuasive because Applicants are arguing limitations that are not present in the claims.

8. Claims 24-26, 28-30 and 43 are rejected under 35 U.S.C. § 103 as being unpatentable over Geisterfer-Lowrance in view of Mullis.

Geisterfer-Lowrance teaches methods for detecting the presence of mutations associated with hypertrophic cardiomyopathy wherein the methods comprise detecting the presence of point mutations in the *B*-MHC nucleic acids by isolating DNA from individuals affected with hypertrophic cardiomyopathy and sequencing the DNA in order to identify the presence of mutations associated with hypertrophic cardiomyopathy (see, e.g., page 1000). In particular, Geisterfer discloses the presence of the missense mutation Arg403Gln and the association of this mutation with individuals having hypertrophic cardiomyopathy. The reference (see abstract) states that the "(I)dentification of two unique mutations within cardiac MHC genes in all individuals with FHC from two unrelated families demonstrates that defects in the cardiac MHC genes can cause this disease". Geisterfer-Lowrance does not teach amplifying the sample *B*-MHC nucleic acid prior to determining the sequence of the DNA.

Mullis teaches methods for amplifying nucleic acids by the method of PCR and applies this methodology to assays to detect the presence of point mutations in nucleic acids associated with genetic diseases (see, e.g. col. 2, and 18). Mullis also teaches amplifying nucleic acids by PCR prior to sequencing (see column 36). The reference states that PCR provides the advantages of increasing the quantity of the target nucleic acid and thereby increases the sensitivity of nucleic acid detection and characterization assays. Mullis further teaches that the presence of mutations associated with a disease can be detected in a sample RNA by first reverse transcribing the RNA

to DNA, amplifying the DNA by PCR and then analyzing the amplified DNA for the presence of disease associated mutations.

Accordingly, it would have been obvious to one of ordinary skill in the art at the time the invention was made to have modified the method of Geisterfer-Lowrance so as to have amplified the B-MHC nucleic acids prior to sequence analysis in order to have increased the quantity of the target DNA and thereby to have increased the overall sensitivity of the detection of hypertrophic cardiomyopathy associated point mutations in the *B*-MHC nucleic acids.

In the response of Paper No. 27, Applicants argue that there is no expectation of success for formulating the claimed methods and state that there is no suggestion in the cited references for formulating a method for detecting mutations associated with HC. These arguments are not persuasive because the claims are not directed to methods of diagnosis but rather only to methods for identifying mutations in the *B*-MHC gene. Applicants arguments that the prior art must teach that a specific mutation is unequivocally associated with HC is inappropriate because the claims do not require diagnosis of HC. The claims are drawn only to methods to detect a mutation associated with HC. Geisterfer-Lowrance provides the motivation and reasonable expectation for identifying such mutations in other nucleic acid samples because the reference provides the methodology for detecting such mutations and it would have been well within the skill of the art to have practiced these conventional methods to effectively accomplish the analysis of nucleic acids for mutations. Furthermore, the reference provides the motivation to analyze additional samples for the stated mutations because the reference teaches that further assays should be performed to determine if the mutation is present in other families and states that use of genetic

probes to MHC mutations will be important in facilitating our understanding of the function of MHC and the causes of HC. Accordingly, the cited prior art suggests and provides a high expectation of success of employing methods for the detection of mutations in MHC. Applicants provide details of the research that they have performed to detect point mutations in MHC that are correlated with hypertrophic cardiopathy. It is stated that only with this extensive analysis that "one would have a reasonable expectation of success in facilitating diagnosis of HC in subjects unrelated to Family A by detecting mutations in the *B* cardiac myosin heavy chain gene. This argument is not persuasive because the claims are not limited to "methods for diagnosis of HC". Rather, the claims are drawn to methods for identifying a mutation associated with HCM and methods for detecting the presence of a target sequence in genomic DNA. Since the combined prior art teaches a method for effectively detecting a mutation in the MHC DNA, wherein the mutation has the property of being a hypertrophic cardiomyopathy-associated mutation, and suggests employing this methodology to detect the presence of this mutation in other individuals, the prior art when considered as a whole leads the ordinary artisan to a method for detection of a HCM associated mutation.

9. Claim 27 is rejected under 35 U.S.C. § 103 as being unpatentable over Geisterfer-Lowrance in view of Almoguera.

Geisterfer-Lowrance teaches methods for detecting the presence of mutations associated with hypertrophic cardiomyopathy wherein the methods comprise detecting the presence of point mutations in the *B*-MHC nucleic acids by isolating DNA from individuals affected with hypertrophic cardiomyopathy and sequencing the DNA in order to identify the presence of

mutations associated with hypertrophic cardiomyopathy (see, e.g., page 1000). In particular, Geisterfer discloses the presence of the missense mutation Arg403Gln and the association of this mutation with individuals having hypertrophic cardiomyopathy. Geisterfer-Lowrance does not teach detecting point mutations associated with hypertrophic cardiomyopathy by first amplifying sample *B*-MHC nucleic acids and performing a RNase protection assay.

Almoguera teaches methods for identifying gene mutations associated with genetically inherited diseases wherein the methods comprise amplifying a DNA sequence by PCR, combining the amplified DNA with a labeled RNA probe in order to form a RNA/DNA hybrid, and performing an RNase protection assay wherein cleavage of the RNA/DNA at regions that are not hybridized as indicative of the presence of a disease associated mutation (see, for example, pages 39-41). In particular, the assay identifies single-base substitutions or point mutations which are considered to be "small alterations" in the DNA. Almoguera states that this provides a very rapid, efficient and sensitive means for detecting the presence of point mutations associated with diseases.

In view of the disclosure of Almoguera, it would have been obvious to one of ordinary skill in the art at the time the invention was made to have modified the method of Geisterfer-Lowrance so as to have detected the mutations associated with hypertrophic myocardiopathy in *B*-MHC nucleic acids by amplifying the nucleic acids by PCR and detecting the presence of mutations by performing an RNase protection assay using a labeled RNA probe in order to have achieved the expected advantages of providing a more rapid, efficient, and sensitive assay for the detection of hypertrophic myocardiopathy associated mutations in *B*-MHC nucleic acids.

In the response of Paper No. 27, Applicants traversed this rejection for the reasons stated above. Accordingly, the response to those arguments apply equally to the present grounds of rejection.

THIS ACTION IS MADE FINAL. Applicant is reminded of the extension of time policy as set forth in 37 CFR 1.136(a).

A shortened statutory period for reply to this final action is set to expire THREE MONTHS from the mailing date of this action. In the event a first reply is filed within TWO MONTHS of the mailing date of this final action and the advisory action is not mailed until after the end of the THREE-MONTH shortened statutory period, then the shortened statutory period will expire on the date the advisory action is mailed, and any extension fee pursuant to 37 CFR 1.136(a) will be calculated from the mailing date of the advisory action. In no event, however, will the statutory period for reply expire later than SIX MONTHS from the mailing date of this final action.

Any inquiry concerning this communication or earlier communications from the examiner should be directed to Carla Myers whose telephone number is (703) 308-2199. The examiner can normally be reached on Monday-Thursday from 6:30 AM-5:00 PM.

If attempts to reach the examiner by telephone are unsuccessful, the examiner's supervisor, W. Gary Jones, can be reached on (703)-308-1152. The fax number for the Technology Center is (703)-305-3014 or (703)-305-4242.

Any inquiry of a general nature or relating to the status of this application should be directed to the receptionist whose telephone number is (703) 308-0196.

Carla Myers
March 23, 2001

Carla Myers
CARLA J. MYERS
PRIMARY EXAMINER